Echo-Planar and Conventional Imaging of Signal Attenuation in Skeletal Muscle during Arterial Compression

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PURPOSE:

In the present study, we observe, with the use of gradient-echo and spin-echo echo-planar imaging (EPI) and conventional imaging, the modulation of signal from resting muscle tissue in the human forearm during arterial compression.

INTRODUCTION:

During exercise, it has been observed that muscle tissue T2 and T2* increase (1), yet the T2 of blood returning from the active muscle decreases (2). The increases in T2 and T2* of the muscle tissue have been attributed to an increase in water content associated with exercise-induced increased blood perfusion, and the decrease in T2 of blood has been attributed to an increase in paramagnetic deoxyhemoglobin concentration associated with the exercise-induced increase in oxygen extraction fraction. In cerebral gradient-echo imaging, it has been found that, during arterial occlusion, an increase in deoxyhemoglobin in the blood increases the bulk susceptibility differential between vessels and tissue, thus increasing intravoxel dephasing and attenuating the signal (3). It is known that the concentrations of both deoxyhemoglobin and deoxymyoglobin increase during arterial compression of skeletal muscle (4). Therefore, the effects of an increase in deoxyhemoglobin and possibly compartmentalized deoxymyoglobin rather than an exercise-induced increase in water volume should dominate the observed change in signal.

METHOD:

Arterial compression was obtained by placing a sphygmomanometer cuff on the arm and inflating to above 300mmHg. Conventional imaging and EPI were performed on a clinical GE 1.5-T Signa system using a 10.4 cm i.d. three-axis local gradient coil. Conventional spin-echo and gradient-echo images (TR=500ms, TE=45ms) having an 8cm FOV, 15mm slice thickness, and 128 x 256 resolution were obtained of a cross-section of a human forearm before and between minutes 3 and 5 of arterial compression. Blipped, gradient-echo and spin-echo EPI pulse sequences, were also used. Acquisition time was 40 ms to acquire each 64 x 64 image. The FOV, slice thickness and TE were the same as above. A series of 128 sequential axial images was obtained using an inter-scan delay of 3 s. During the time course series, arterial compression was initiated at image 20 and discontinued at image 90.

RESULTS:

After the onset of compression, signal attenuation of 9% to 21% was observed using the gradient-echo EPI sequence and signal attenuation of 5% to 9% was observed using the spin-echo EPI sequence. Upon release of the compression, an overshoot in signal, corresponding to reactive hyperemia, is observed to be larger and more immediate in the gradient-echo sequence than in the spin-echo sequence. Figure 1 is a typical experiment comparing the muscle tissue signal attenuation in both sequences.

Using a conventional gradient-echo sequence, signal attenuation of 14.3% ± 3% was observed in muscle tissue during occlusion. Also, darkening around resolved vessels increased in a manner similar to that found in deoxygenated rat cerebral vessels (5) No significant signal change was observed in muscle tissue during occlusion using a conventional spin-echo sequence.

DISCUSSION:

During arterial compression, blood volume does not change significantly and oxygenation is known to decrease. It is hypothesized that since attenuation is observed in both spin-echo and gradient-echo EPI, and since the gradient-echo attenuation is more pronounced, susceptibility-related intravoxel dephasing in the intermediate exchange regime demonstrated (6). Furthermore, given the very small perfusion fraction of blood in resting muscle and the very large observed attenuation, it appears that an increase in deoxymyoglobin concentration in muscle may additionally contribute to the susceptibility-related attenuation. For changes in paramagnetic deoxymyoglobin concentration to contribute to the changes in signal, myoglobin must be sufficiently compartmentalized so that microscopic field inhomogeneities are created upon deoxygenation. Compartmentalization of myoglobin remains to be proven.

The observed delay in the spin-echo EPI attenuation and overshoot indicates that the primary source of change in T2 dephasing is delayed from the primary source of change in T2* dephasing.

Conventional imaging appears less sensitive than EPI to subtle signal changes that occur upon arterial compression. The reason for this may be due to system instabilities or physiologic motion during the scan.

CONCLUSION:

Signal attenuation in spin-echo and gradient-echo EPI and conventional gradient-echo imaging is observed during arterial occlusion, and is attributed to deoxygenation in the resting muscle. This technique may prove to be a powerful new tool in the investigation of oxygen delivery and hemodynamics in normal or pathological states such as hypertension. Further study of the timing and magnitude of the EPI signal modulation may reveal new information about the muscle hemodynamic response and/or the nature of deoxymyoglobin compartmentalization.

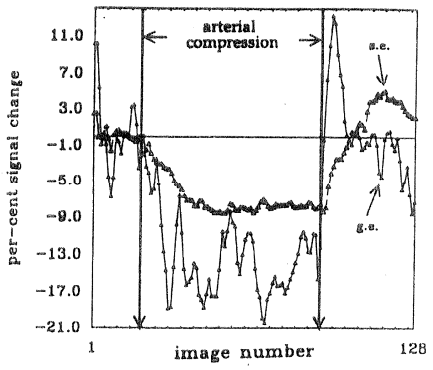

Figure 1

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